Evolutionary Function of Enhanced Probability of Embryo Sex Being Set as Male in Humans and Hypothesis Concerning Mechanism Through Which This Bias in Probability Is Introduced

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Simon Edwards
Research Acceleration Initiative

Introduction

It has long been recognized that there is a greater than 50% probability (about 53%) of a given human embryo becoming male rather than female. Furthermore, male children are less likely to succumb to childhood illnesses and become closer to 54% of the population for the 10-14 age bracket according to available statistical data. From there, females switch over to become the group more likely to survive until finally, at age 60, women begin to outnumber men, living; on average; four years longer.

This phenomenon has not be properly studied from either an evolutionary or from a functional biological perspective.

Abstract

The first question this author proposes to answer is the one of what evolutionary benefits having a greater number of males than females during the period of male fertility confers to the human species and what evolutionary benefits may be associated with increased longevity for females. It is this author's contention that a slightly greater number of males than females creates a useful sort of evolutionary pressure as a result of greater competition between males and a greater number of options for the females of the species. The mathematical average consequence of this bias in the ratio of males to females is that males must be, generally speaking, in the 53rd percentile of "fitness" (however one might choose to define it,) in order to have a greater than equal likelihood of passing on their genes. This contention is supported by the established understanding that human females, generally speaking, make the ultimate decisions concerning mate selection. The greater than equal likelihood of embryos being set as 'male' has the tendency to improve the quality of offspring in a system in which females make the mating decisions.

Another dimension of this phenomenon is the fact that increased longevity for females enhances survival generally, as supported by previous studies, as elderly females can serve as caregivers for children; something which pays dividends toward the survival of tribes and individuals.

If the increased likelihood of embryos being set as male in the human species provides an evolutionary benefit, the next question one must answer is which exact mechanism of biology is responsible for facilitating this bias. To answer that question, even hypothetically, we must consider statistical observations and

correlations between unique aspects of female biology in order to narrow the possibilities for which process enables such a bias. Open questions include whether this bias, which averages 53%-47% in favor of male conception can vary depending upon circumstances and for how long this bias has existed.

I propose that the most likely candidate mechanism for variably governing the degree of such a bias toward male children is the hormone, estrogen. This notion is, for some, counterintuitive given that most people associate estrogen with female biology.

Individual women have varying baseline levels of estrogen and this hormone is produced in the ovaries; very near to where unfertilized eggs are stored. Fluctuations in the amount of estrogen produced by the ovaries would result in corresponding fluctuations in the amount of estrogen in the endoplasmic reticulum of the egg cells. I propose that it is the level of estrogen within the endoplasmic reticulum of egg cells at the time of conception which determines the probability of the embryo being set as either male or female. However, rather than a higher quantity of estrogen making sex assignment as female more likely, I propose that the opposite is true for reasons of estrogen's effect upon gene expression on the Y chromosome.

When the Y chromosomal material of the male reproductive cell is exposed to the estrogen native to the endoplasmic reticulum of the egg just after conception, its expression of mRNA signaling is amplified. The X chromosome introduced by the male would not be affected one way or the other by the presence of the estrogen. If Y is stimulated by the ambient estrogen, it could be expected to attach and supplant one of the mother's X chromosomes during that step in the process of the establishment of the embryo's DNA. Y must cut away one of the mother's X chromosomes and supplant it and it does this by secreting proteins which function to separate the two maternal X chromosomes from one another and subsequently secreting others which enable its own binding to the remaining X once within close proximity.

Importantly, when Y fails to be met with estrogen, there is nothing to signal to Y that it should become active. Because Y is shorter in length than X, the volume of signaling chemicals/proteins it secretes is far lower, by nature, without some artificial stimulus. Estrogen is that stimulus. A precisely calibrated level of ambient estrogen within an egg cell serves somewhat like a precisely calibrated step-stool upon which the Y chromosome may stand so that it is, in terms of chemical signaling, made to be the "same height as" X, at least in terms of chemical/mRNA signaling activity.

Therefore, a woman with lower estrogen levels either for reason that she has a naturally low baseline level or due to advancing age could be expected to be more likely to produce female children whilst a woman with high estrogen levels would be statistically more likely to produce a series of sons and fewer daughters.

As levels of hormones such as estrogen can vary within even the same individual over time and depending upon circumstance and baseline levels with females may fluctuate over a period of many generations depending upon resource-availability. Conditions of abundance might be the factor which drives the bias of the female reproductive system toward producing more males through the enhancement of estrogen production. During periods of abundance, a female might wish to be more selective when choosing a mate. The availability of food consistently over a young woman's youth could be expected to provoke increased production of estrogen over time. If food is scarce, hormone production could be expected to be hampered. This would, in turn, shift the probability of male or female sex assignment to a ratio closer to parity or could even result in a swing in the opposite direction in which more children are born female than male.

Conclusion

This hypothesis, may, incidentally, offer some explanation for the tendency toward the shortening of the Y chromosome which has been noted in some observational studies. The chief practical ramification of an increasingly short Y chromosome, it would seem, is the conferral of a greater degree of control by the male to the female over the probability ratio of the sex of offspring. If all of the aforementioned premises are experimentally verified, the further shortening of the Y chromosome might result result in an increasing statistical skew in favor of male births over female births and a consequent increase in selective forces against unfit males. Should food resources continue to be abundant for the human species, it would seem likely that this trend toward Y chromosomal shortening (and thus, a greater number of men than women) should continue.